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Intra-aortic Balloon Counterpulsation in the Treatment of Cardiogenic Shock: Hemodynamic Effects and Clinical Challenges CME

Author: Spyridon D. Moulopoulos, MD

Complete author affiliations and disclosures are at the end of this activity.

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Target Audience

This activity is intended for physicians.

Goal

The goal of this activity is to explore acute conditions, including cardiogenic shock, in which mechanical circulatory support through IABC can reduce morbidity and mortality and/or serve as a bridge to cardiac transplantation.

Learning Objectives

On completion of this continuing medical education offering, participants will be able to:

1. Identify the public-health dimensions of, and risk/prognostic factors for, cardiogenic shock after AMI.
2. Recognize the need for counterpulsation and the trend toward early insertion of the device for high-risk AMI patients undergoing revascularization.
3. Summarize the indications, contraindications, and balloon-catheter placement techniques for counterpulsation.
4. Explain the hemodynamic benefits of IABC.

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Contents of This CME Activity

This activity is not sanctioned by, nor a part of, the First World Conference on Intra-aortic Balloon Counterpulsation (Chairman, Spyridon D. Moulopoulos, MD). Click here for more information on this Medscape program.

Intraaortic Balloon Counterpulsation in the Treatment of Cardiogenic Shock: Hemodynamic Effects and Clinical Challenges

Chairman's Foreword

"Many useful matters are yet treasured up in the bosom of Nature, out of the common track of our imagination, and still undiscovered, and which will doubtless be brought to light in the course and lapse of years, as others have been before them." -- Francis Bacon

Although his observation comes to us from several centuries ago, many in the scientific and medical communities believe that Francis Bacon's comment (above) is as applicable today as it was in his time. And thus it was with this thought in mind that I brought together a distinguished international convocation to discuss one of the most crucial and yet widely misunderstood medical therapies of our time -- intra-aortic balloon counterpulsation (IABC)

As one of the original contributors to the development of IABC more than 35 years ago, I have been particularly concerned that the clinical potential of IABC has yet to be realized. In recent years, IABC has been the most frequently used of all available heart-assist devices, and yet it is widely believed that counterpulsation therapy is vastly underutilized as a clinical strategy to manage low cardiac output due to left ventricular (LV) dysfunction.

This observation is based on the fact that many patients with, or at risk of, cardiogenic shock are not receiving the potential health benefits of this safe and effective form of circulatory support. This failure of the medical community to avail itself of the full range of possible applications of IABC may be due to the unavailability of equipment, to a lack of acquaintance with the possibilities, or perhaps to fear of the complications that were reported early in the era of counterpulsation. But more than anything else, this failure to use this technique in all of its proper indications appears to be based on the prevailing idea that IABC is destined only for those cases where every other type of treatment has

failed.

Therefore, given this important lack of understanding, it seemed appropriate to convene a meeting of leading experts and practitioners to reevaluate the use and application of IABC. This meeting, with the participation of eminent colleagues, old and new pioneers in the field, was brought together to formulate the proper indications, propagate recent knowledge, and point to new directions of research.

Accordingly, with the gracious cooperation and generous assistance of the Datascope Corporation, and working in concert with Professors Demopoulos and Stamatelopoulos from Athens University, as well as Associate Professor Nanas and Assistant Professor Lekakis, I organized this meeting, the First World Conference on Intra-aortic Balloon Counterpulsation. Consistent with the goal of disseminating the aggregate wisdom of this learned group to interventional cardiologists, thoracic surgeons, intensivists, and other practitioners, I chose the city of Athena, the goddess of wisdom; the birthplace of Plato, the founder of Western philosophy; and a leading center for innovation in the development of vital heart-assist methods.

On behalf of the organizing committee, I would like to express our thanks to the directors of Athens University for the time and effort they devoted to the organization of the meeting, and also to the Datascope Corporation for their generous support of this meeting.

Chairman's Introduction

In this part of the Mediterranean world, the appreciation of surgeons dates back almost 3000 years. Homer, for example, speaks of Machaon, the surgeon-in-chief during the Trojan War, as a man worth several other men put together -- although it may be worth noting in passing that in treating war wounds, Machaon's patients had a mortality rate of 76%. And the other son of Aesculapius, Podalirius, who was an internist, was cited by Homer as the physician who "knows the unseen and heals the incurable."

Moving forward to our times, at the midpoint of the 20th century a group of daring investigators began pursuing the dream of a mechanical pump to replace the irreparably sick human heart. They were inspired by Kolff's successful creation of a dialysis machine to replace the ailing kidneys and encouraged by the application of Gibbons's extracorporeal heart and lung machine.

In the search for a way to assist the failing heart, many surgeons dedicated time and effort. I mention only De Bakey's, Kantrowitz's, and Dennis's groups, although there were many others. However, it was a physician, not a surgeon, who spent his whole life trying to build an artificial heart: our honorary president and doctor *honoris causa* of this university, Willem Kolff. When he started with this project, the obstacles were immense and, to many, including some of his coworkers, insurmountable. Under these circumstances, a more modest target appeared closer to the capabilities of the technologies then available, namely, assistance to the heart instead of total replacement.

The first approach was assistance in parallel to the heart, which was achieved by several bypass techniques, most of them requiring surgical thoracotomy. Assistance in series was attempted by Harken with an extracorporeal pump, which took blood from the aorta in systole and reinjected it during diastole. This was the principle of "counterpulsation" that ultimately found a more convenient application in the intra-aortic balloon, as will be discussed below in this report from the First World Conference on Intra-aortic Balloon Counterpulsation.

To conclude this introduction, however, I merely point out that today, almost 40 years later, IABC is the most widely applied assist technique. The reasons for this success are multiple. It is an assist technique that is incorporated in series, is minimally invasive, does not handle blood outside the body, does not cause hemolysis, and can help both the surgeon and the cardiologist in treating otherwise untreatable heart disease. I would also suggest that this technique showed the way to other methods that use balloons inside the circulatory system by demonstrating the possibility of such interventions.

Thus, with this perspective in mind, and with appreciation again for the contributions of my distinguished colleagues toward the success of this medical technology and the success of this meeting, we should turn now to consideration of the development of IABC, beginning with an examination of the pathology that it was created to treat.

Development of Intra-a rtic Balloon Assistance in Hist rical Perspectiv

At the dawn of the new millennium, IABC therapy -- a form of supplementary circulatory assistance that will also be termed simply counterpulsation in this review -- has been in use for nearly 40 years. Willem Kolff, Stephen Topaz, and I conceived this procedure at the Cleveland Clinic in 1961,[1] and Kantrowitz applied it to patients in 1967.[2]

Our tests, which were conducted in a mock-circulation system, enabled us to evaluate parameters for the carbon-dioxide-filled catheter-mounted balloon, including pressure within the balloon, balloon volume, length of polyurethane tubing to be passed into the aorta, and stroke duration. Experiments in dead dogs demonstrated that a pressure of 80/60 mm Hg could be developed using rapid infusion of saline into the arterial system, but only if the initial pressure equaled (or exceeded) 40 mm Hg.

Next, testing in live anesthetized dogs assisted in optimizing the delay time of the pump stroke to within 20 ms of the dicrotic notch; if the entire pump stroke was not complete before the next ventricular systole, the improper delay time caused the pump stroke to occur during systole -- thereby actually increasing the load on the ventricle.[1]

Finally, using cineangiography in human cadavers, we demonstrated that IABC caused substantial movement of radio-opaque dye from the ascending aorta into both peripheral and coronary arteries despite cardiac arrest.[1] We concluded that this method, which enabled diastolic augmentation and reduced end-diastolic pressure, afforded a number of potential clinical advantages: It was relatively simple, required cannulation of only 1 vessel, and did not necessitate either priming of the device with blood or extracorporeal handling of blood.

Subsequently, Kantrowitz's group reported the first clinical experience with IABC.[2] However, as mentioned by O'Rourke in an editorial accompanying a report on IABC for intractable cardiogenic shock,[3] Kantrowitz's favorable early findings, and particularly the definitions of cardiogenic shock, came under intense scrutiny and criticism. Unfortunately, this led to the adoption, in certain institutions, of more stringent criteria, which led to enrollment of patients with exceedingly poor prognoses and, with this trend, poorer clinical outcomes. In concert with unfavorable survival data, which discouraged many practitioners, implementation of the new shock criteria was also difficult because many patients and/or healthcare personnel objected to the "drawn-out routine" of preliminary catheterization, balloon insertion, coronary arteriography, weaning, then surgery on the coronary arteries.[3]

Perhaps partly as a result, IABC lost favor in many circles during the 1970s. However, during this period, advances in surgical interventions for acute myocardial infarction (AMI) brought new identification of correctable, mechanical sources of myocardial disruption, including papillary-muscle rupture, acute septal perforation, and hemopericardium, some of which were once construed as consequences rather than causes of cardiogenic shock.[3] Throughout the 1980s and 1990s -- and up to the present time -- the study and practice of IABC and other forms of left ventricular (LV) assist continued in Athens, at Athens University Alexandra Hospital. Studies by Dr. Nanas, Dr. Stamatelopoulos, Dr. Anthopoulos, Dr. Charitos, myself, and others demonstrated that IABC enhanced clinical outcomes (eg, survival) in patients with severe or intractable cardiogenic shock,[3-8] congestive heart failure,[9] or mitral regurgitation.[10]

Cardiogenic Shock: Historical, Demographic, and Clinical Perspectives

First characterized in 1942, cardiogenic shock remains a formidable clinical and public-health challenge. Indeed, the classic definition of this low-output cardiac syndrome is essentially unaltered 6 decades after the term *cardiogenic shock* was coined. The patient in cardiogenic shock "presents signs of a decreased peripheral blood flow with diminished or no radial pulse, cold extremities, narrowed pulse pressure, and a relatively well maintained diastolic pressure," according to a case series published during the World War II.

Nearly 60 years later, Hasdai and coworkers[11] confirmed that the clinical manifestations of cardiogenic shock first identified in the early case series, including low urine output, oliguria, altered sensorium, and cold, clammy skin, portend a poor prognosis. Absent at presentation of AMI in up to 90% of cases,[12,13] these symptoms of cardiogenic shock are associated with an increased risk of mortality. Logistic-regression models that incorporated hemodynamic and demographic variables, among others, demonstrated that AMI patients with oliguria were at 2.25 times higher 30-day mortality risk. Individuals presenting with either altered sensorium or cold, clammy skin were at 1.7 times the risk of dying as their counterparts who did not present with such symptoms.

The leading cause of mortality among patients with AMI treated in US hospitals,[14] cardiogenic shock is a major clinical and public-health challenge. According to a longitudinal population study involving more than 9000 AMI patients,[15] the incidence of cardiogenic shock remained relatively stable over the past 3 decades, at about 7% annually.

Further, despite a striking decrease in coronary artery disease (CAD) mortality from 1980 to 1990,[16] in-hospital case fatality rates for cardiogenic shock remained above 70% among nearly 5000 patients admitted with AMI to hospitals in the Worcester, Massachusetts metropolitan area from 1975 to 1988.[17] By recent estimates,[14] nearly 60% of all hospital deaths can be attributed to cardiogenic shock, which leads to about 70,000 deaths each year in patients hospitalized for AMI.[18]

Role of IABC in Supporting the Failing Heart

As a form of mechanical circulatory assistance, counterpulsation can substantially enhance prognosis in cardiogenic shock. Aortic counterpulsation assists the failing heart after AMI, unstable angina (UA), and/or cardiac surgery by inflating during diastole, increasing diastolic pressure and, with it, cardiac perfusion. Deflation of the balloon during systole (immediately before the next valve opening) effectively unloads the heart, enabling the left ventricle to eject against decreased impedance. The foregoing effects promote myocardial energy balance in the failing heart, sharply diminishing myocardial oxygen consumption. Finally, counterpulsation attenuates left-to-right shunting across a ventricular septal defect (after AMI) or regurgitant flow across mitral valve defects by decreasing ventricular pressures and volumes.

The hemodynamic benefits of counterpulsation therapy can substantially reduce morbidity and mortality for patients in need of mechanical circulatory support after AMI and/or cardiac surgery. For example, mortality was 57% in the Global Utilization of Streptokinase and Tissue-Plasminogen Activator for Occluded Coronary Arteries - I (GUSTO-1) trial,[14] in which the frequency of IABC use was 24%. On the other hand, mortality attributable to cardiogenic shock reached 70% in 2 other major thrombolysis trials with less frequent institution of IABC: the Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico (GISSI)[13] and the International Study.[19]

Further support for the role of IABC in enhancing survival in AMI complicated by cardiogenic shock derives from 2 smaller, observational studies, First, Goldberg and associates[15] determined that IABC use was more than twice as common in survivors (33.5%) than in patients who died from cardiogenic shock (14.7%; $P < .001$). Similar rates of treatment with thrombolysis and percutaneous transluminal coronary angioplasty (PTCA) were reported in patients who survived cardiogenic shock, as compared with only 17.9% and 9.0% in nonsurvivors ($P < .001$ for both comparisons).[20]

Second, the overall in-hospital mortality rate was only 53% in a small population with an IABC treatment rate of 50%: 52% of patients who received such therapy survived cardiogenic shock complicating AMI, as compared with 43% of those who died ($P = .23$).[20] The mortality rate declined to 38% among patients treated with both mechanical revascularization and IABC.

Trends in Current Uses of IABC

Although IABC was, for many years, implemented as a response to cardiogenic shock, a more enlightened, proactive approach is emerging -- particularly when performing PCI or cardiotomy in high-risk patients. In the Thrombolysis and Angioplasty in Myocardial Infarction (TAMI) trial, 75% of IABCs were inserted either before or during cardiac catheterization in 810 consecutive patients initially treated with thrombolysis.

Why is early balloon placement clinically important? Not only do the majority of patients in Killip class IV shock die within the first 48 hours, but available interventions to prevent or treat cardiogenic shock are insufficient without adjunctive IABC. For instance, the GISSI-1 trial[13] demonstrated that thrombolysis confers no clinical benefit if cardiogenic shock is already present.

In a retrospective analysis of nearly 5000 cases of IABC support at the Massachusetts General Hospital (MGH) since 1968, Torchiana and colleagues[21] determined that preoperative balloon counterpulsation reduced in-hospital mortality after cardiac surgery by nearly 3 times: to 13.6% from 35.7% for intraoperative placement; the authors contended that "an institutional bias toward preoperative use of the balloon pump appears to be associated with improved outcomes."[21]

These data are consistent with findings indicating that, in contradistinction to postoperative counterpulsation, preoperative IABC tended to diminish in-hospital mortality and morbidity, including postoperative cardiac arrest and

overall complications. Late mortality at this institution was frequently attributed to persistent HF or sudden cardiac arrest. Other trends in IABC use at MGH included a 50% relative risk reduction in mortality (from 41% to 20%) over the 30-year study interval, increasing frequencies of counterpulsation procedures for management of ischemic conditions, and advancing mean patient age.[21]

Another retrospective chart analysis[22] demonstrated the survival benefit conferred by IABC support in the setting of cardiogenic shock after thrombolysis for AMI. In the community hospitals studied, patients who received counterpulsation support exhibited a 93% in-hospital survival rate as compared with 37% without IABC ($P = .0002$). Death occurred within 7 hours in the usual-care group as against 24 hours in the IABC group.

According to preclinical work by Gurbel and colleagues,[23] diastolic augmentation through IABC accelerated recombinant tissue plasminogen activator (rt-PA) reperfusion in dogs with experimentally induced critical LAD lesions: from nearly 40 minutes in animals receiving an intravenous, front-loaded rt-PA regimen to 13 minutes in those treated adjunctively with counterpulsation. These results were ascribed to beneficial effects of the augmented DBP wave form on the LAD thrombus.

Using a canine model of coronary thrombosis, Prewitt and coworkers[24] determined that experimentally induced systolic hypotension (BP < 90 mm Hg) was associated with a sharply decreased rate of rt-PA thrombolysis. On the other hand, counterpulsation increased the rate of clot lysis by up to 1169% (mean, 83%).

Geographical and institutional variability in IABC use is marked. Even within the state of Massachusetts,[25] counterpulsation rates ranged from 7.8% to 20.8% of coronary artery bypass grafting (CABG). Apart from cardiogenic shock per se, independent risk factors for adjunctive IABC with CABG included same-admission PTCA, prior CABG, CHF, recent MI, and cardiac arrest. As compared with their European counterparts, US investigators in the GUSTO trial[26] were 5 times as likely to use IABC (35% vs 7%; $P < .001$).

The foregoing favorable pattern was consistent with an overall trend toward more aggressive PCI in the United States, including significantly higher rates of cardiac catheterization, CABG, and PTCA, as well as significantly more frequent use of beta-blockers and inotropic agents. Institution of IABC was among several independent risk factors significantly correlated (inversely) with 30-day mortality in patients with cardiogenic shock in the GUSTO trial. Longer time to treatment was also a significant prognostic factor for mortality in this study.[26]

Current Indications

Although American Heart Association guidelines drafted in part by Dr. Ohman are imminently awaited, at present the following clinical indications for IABC are widely recognized:[27]

- pump failure or cardiogenic shock as indicated by hemodynamic instability (BP < 90 mm Hg, cardiac index < 2, pulmonary capillary wedge pressure > 18 mm Hg, ST elevation) refractory to volume optimization and inotropic support
- cardiac surgical patients at risk for hemodynamic decompensation
- severe left-main disease
- ischemic changes during hypotensive episodes (anesthesia)
- hemodynamic or ECG instability in catheterization laboratory (ie, "cath lab")
- ventricular arrhythmia unresponsive to conventional treatment
- cardiac failure due to CAD complications resistant to inotropes and diuresis
- LV aneurysm
- acute mitral insufficiency

- mechanical complications of AMI (eg, acute mitral regurgitation)
- postinfarction ventricular-septal rupture
- hemodynamic/ECG instability during early surgical phase [pre-cardiopulmonary bypass]
- failure to wean from cardiopulmonary bypass
- bridge to cardiac transplantation.

Insertion Technique

Among approaches utilized to insert the IABC balloon catheter, the Seldinger technique involves retrograde insertion through the femoral artery (with or without a sheath). Recent years have witnessed a marked trend toward percutaneous rather than surgical insertion of the device, improvements in hardware and other equipment, and the use of percutaneous closure of the artery. During the past 30 years, surgical insertion techniques, which involve surgically accessing the common femoral artery, iliac artery, ascending aorta, subclavian artery, or axillary artery, have also been used.[21,27]

Introduction of cath-lab transesophageal echocardiography (TEE) or roentgenography (in the ICU) enables the physician to check the placement of the IABC under fluoroscopic control. Transesophageal echocardiography can be used to rule out causes of cardiac failure for which IABC may not be needed, such as hypovolemia.[28] Moreover, in certain cases, TEE helps to confirm placement of the guide wire in the thoracic aorta before larger-bore dilators and the balloon catheter are inserted. Once the balloon catheter has been inserted, TEE can assist in confirming proper positioning of the catheter tip. TEE can also expedite detection of acute aortic dissection, a potential complication of counterpulsation balloon placement.[29]

Major Cardiogenic-Shock Registries

SHOCK Registry

The SHould we emergently revascularize Occluded Coronaries for cardiogenic shocK? (SHOCK) registry was conducted to compare medical stabilization with surgical or percutaneous revascularization for the treatment of cardiogenic shock. An early analysis of the SHOCK registry data suggested that patients selected for revascularization exhibited lower mortality rates, irrespective of whether they received revascularization.[30] This finding led to concerns about selection bias.

This concern was largely resolved through an important randomized, clinical trial conducted recently by Hochman and colleagues.[31] In this study, patients with suspected cardiogenic shock as a result of AMI were randomly assigned to either thrombolysis or revascularization with PTCA or CABG within 6 hours of randomization. Counterpulsation was recommended for all patients.

Demographics of the 302 randomized patients included: mean age of 65.8 years, 67.9% male, 75.7% white, 46.3% diagnosed with hypertension, and 31.1% diagnosed with diabetes. Median time to onset of cardiogenic shock was 5.6 hours after AMI. IABC was used in approximately 86% of patients in both intervention groups.

Overall mortality rates at 30 days were not statistically different in the 2 study arms (46.7% for the revascularization group and 56.0% for thrombolysis; $P = .11$). At 6 months, however, mortality was significantly lower in the group receiving revascularization (50.3% vs 63.1%; $P = .027$). In subgroup analyses, patients 75 years of age or older showed greater benefit with thrombolysis than revascularization at both 30 days and 6 months. Patients without a history of prior MI showed moderately greater benefit with medical therapy, but only at the 30-day assessment. Because of a high rate of IABC use in the control group, patients receiving IABC did not show a significant survival advantage. However, data from the SHOCK registry demonstrated that IABC use substantially deceased mortality.

A more recent analysis of the SHOCK registry data was conducted to evaluate the effects of thrombolytic therapy and IABC on mortality. Sanborn's group[32] found that patients treated with IABC and thrombolytic therapy had a lower mortality than patients treated with either intervention alone or with neither intervention (47% vs 77%; $P < .0001$). In

addition, the researchers noted an association between higher revascularization rates and frequency of IABC use; increased revascularization rates were, in turn, associated with lower in-hospital mortality rates. Finally, the SHOCK Registry also showed that an absence of pulmonary congestion at initial clinical examination of patients with cardiogenic shock was not associated with an enhanced prognosis.[33]

Benchmark Counterpulsation Registry

The ongoing Benchmark Counterpulsation Outcomes Registry is prospectively following the outcomes of patients treated with IABC. Initial results from the registry included 5335 patients from 132 member hospitals. A randomized audit has verified a 95% concordance between source documents and submitted data.

Demographics of the patients entered in the registry included: 66% male, mean age of 66 years, and mean body surface area (BSA) of 1.9 m^2. Diabetes was present in 24% of patients and peripheral vascular disease in 11%. The average duration of IABC was 64.8 hours. As shown in Figure 1, frequent indications for placement of IABC included cardiogenic shock, refractory angina, and the need for support in the cath lab and during weaning from cardiopulmonary bypass. Balloon insertion was most frequently conducted in the catheterization laboratory or operating room.

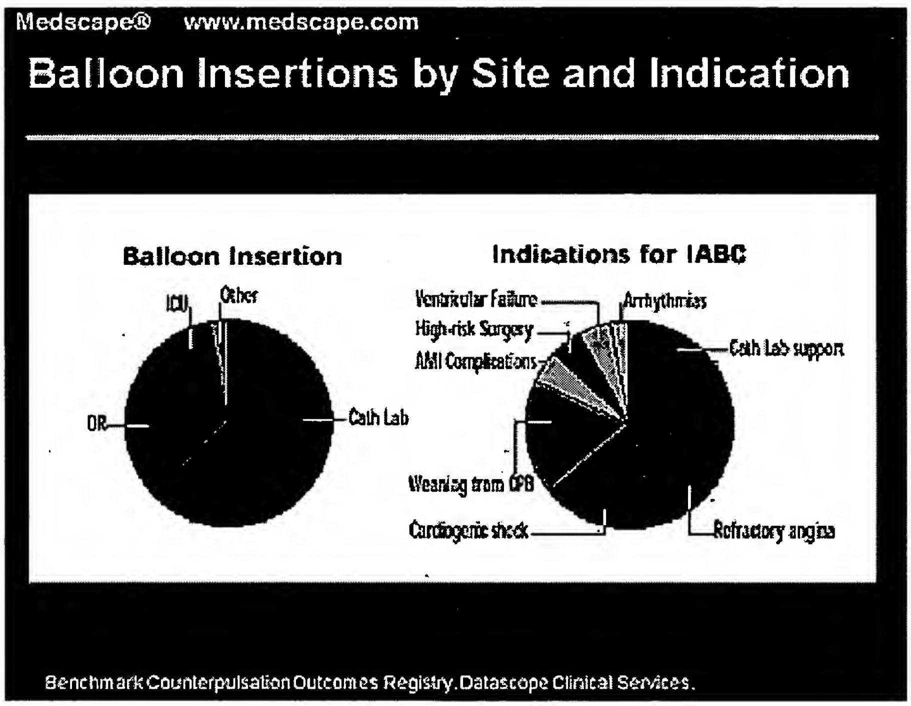

Figure 1. Balloon insertion by site and indication in the Benchmark Counterpulsation Outcomes Registry. Data on file, Datascope Clinical Services.

The in-hospital mortality rate was 20% overall, but the death rate related to IABC was only 0.1%, a discrepancy that reflects the poor prognosis of critically ill patients treated with IABC. The mortality rate while the IABC was in place was 11%. The mean total length of hospital stay was 13.3 days. IABC was unsuccessful in 2.5%, and complications included ischemia, perforation, or severe bleeding in 3.2%, severe bleeding alone in 0.7%, minor ischemia of a limb in 2.0%, and major ischemia of a limb in 1.2%.

Independent patient features that predicted serious complications after IABC (eg, IABC-related death, major ischemia, severe bleeding, balloon perforation) included BSA < 1.65 m^2, (odds ratio [OR], 2.2), female gender (OR, 1.9), and peripheral vascular disease (OR, 1.8). Operator-related risk factors included nonpercutaneous insertion (OR, 2.2) and treatment in a tertiary-care hospital (OR, 1.5).

Since these preliminary data were collected, enrollment has continued at a rate of approximately 450 patients per month, with a total number of enrolled patients of 8858 as of April 1999.

Overview of Randomiz d Clinical Trials in Patients With Cardiog nic Shock

The outcomes of randomized controlled trials involving counterpulsation for cardiogenic shock in the prethrombolysis era were somewhat disappointing. More recently, however, numerous studies involving far larger patient populations have consistently demonstrated the clinical utility of this approach.

In the TAMI trial, the in-hospital mortality rate was 8 times lower (4%) in patients who received IABC than those who did not (32%). As compared with post-AMI patients who received IV alteplase (75 mg/kg) at >/= 12 hours of onset of symptoms, individuals randomized to IABC exhibited significantly enhanced TIMI grade flow rates (74% vs 32% in controls) and minimal lumen diameter (1.6 vs 0.9 mm), as well as diminished residual percent stenosis (42% vs 68%) within infarct-related arteries.[34]

Further, in a trial conducted by Dr. Ohman and associates,[35] 182 patients undergoing cardiac catheterization within 24 hours after MI symptom onset were randomly allocated to treatment with adjunctive IABC (n = 96) or standard care (n = 86), which included thrombolysis (systemic or intracoronary), primary or rescue PTCA, and IV heparin.

Rates of major adverse cardiac events (MACE) -- mortality, stroke, reinfarction, urgent target-vessel revascularization -- were 13% in the IABC group and 24% for standard care ($P < .04$). This composite end point did not include other favorable trends toward significantly lower rates of intermediates -- recurrent ischemia, infarct-related artery (IRA) reocclusion, emergent PTCA -- in the counterpulsation group. Indeed, IRA reocclusion rates were nearly 3 times lower in the counterpulsation group: 8% vs 21% in the standard-care group ($P < .03$). Severe bleeding complications, numbers of transfused units, and need for thrombectomy or vascular repair were similar in the 2 treatment arms.

Finally, in a smaller clinical study, Stomel's group[36] determined that survival was increased by nearly 3-fold ($P = .0049$) with IABC support. Among 64 patients with AMI and cardiogenic shock, mortality rates were 23% in patients who received thrombolysis alone, 28% in those on IABC alone, and 68% in those receiving both thrombolysis and IABC.

Tr atment of High-Risk Patients

AMI

What are the hemodynamic benefits of IABC in high-risk patients? In a clinical trial involving 60 patients with total LAD occlusion, Ishihara and colleagues[37] evaluated the effects of adjunctive IABC support for rescue PTCA after unsuccessful thrombolysis. Counterpulsation not only decreased the occurrence of IRA reocclusion but also promoted recovery of LV function, an effect not typically observed using PTCA alone. When large areas of contractile myocardium are at risk, mechanical support of coronary and systemic circulation is often warranted.

On the basis of serial contrast ventriculograms taken at both the time of reperfusion and at hospital discharge, IABC-treated patients exhibited an increase of 6.8% in LVEF as compared with a change of -2.0% in the rescue-PTCA group (Figure 2; $P < .05$). The LAD reocclusion rate was 10 times lower in the counterpulsation group (2.5%) than among individuals who received rescue PTCA alone (25%; $P < .05$).

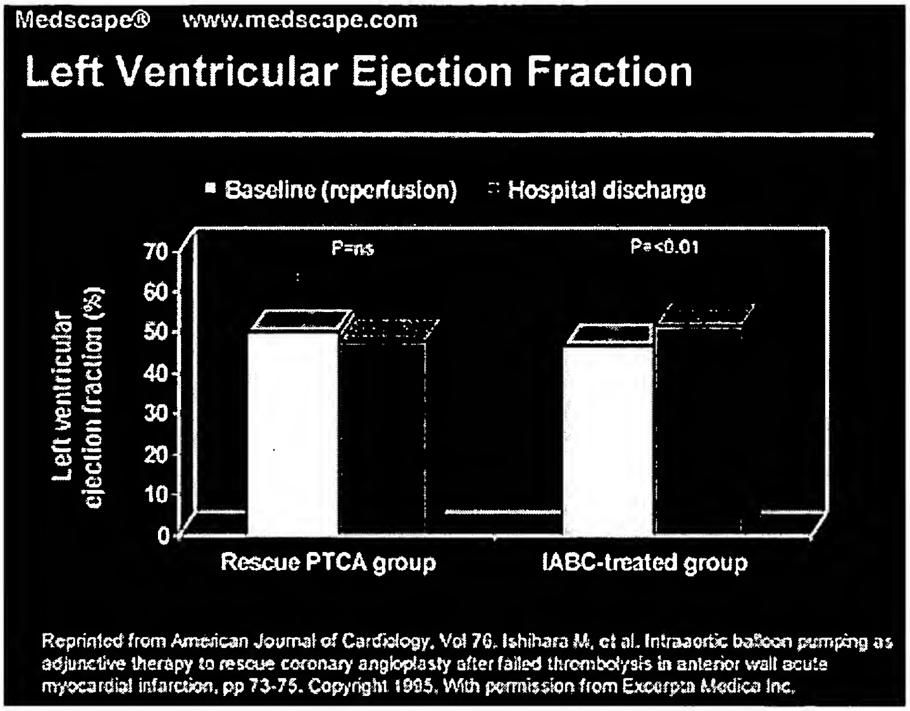

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Figure 2. Changes in left ventricular ejection fraction with rescue PTCA or IABC.[37]

Counterpulsation also clearly increased coronary blood flow (CBF) velocity. In a Doppler ultrasonographic trial involving 19 critically ill patients with clinical indications for IABC (2 of whom had data collected using TEE) Morton Kern and colleagues[38] showed that IABC significantly decreased SBP (LV unloading effect) and augmented DBP without altering the RR interval on ECG.

In addition, IABC therapy significantly enhanced peak and mean phasic CBF velocity by 115% and 67%, respectively, and increased diastolic CBF velocity by 103% over basal values. The latter increase persisted when velocity data were adjusted for HR. Of particular clinical importance in Kern's study, IABC conferred the most pronounced CBF velocity increases in patients with the most severely decompensated hemodynamic status.

On the other hand, prophylactic IABC after PTCA in hemodynamically stable patients is controversial. In the second Primary Angioplasty in Myocardial Infarction (PAMI-II) trial,[39] involving 1100 patients presenting within 12 hours after AMI onset, prophylactic IABC support for primary PTCA conferred no survival benefit. Although the IRA reocclusion rate was also unaffected by IABC, counterpulsation did significantly reduce the rate of unscheduled repeat catheterization and cut the recurrent ischemia rate from 19.6% to 13.3% (P = .08). In trials involving PTCA with intracoronary stent implantation, IABC has not improved clinical outcomes.

Low Preoperative LV Ejection Fraction (LVEF)

Mortality following CABG in patients with LVEF of 0.25 or less is elevated -- up to 14.3%[40] -- despite considerable advances in perioperative pharmacologic support and preservation of functional myocardium. In a retrospective analysis involving 163 consecutive patients with a maximum entry LVEF of 0.25 (mean, 0.22), the mortality rate was more than 4 times lower among 37 individuals who underwent preoperative balloon counterpulsation (2.7%) as compared with those who had CABG alone (11.9%; P = .004). Duration of hospital stay and mean hospital charges were lower in the IABC group, but the differences between IABC patients and those who did not have preoperative insertion of the device were not statistically significant.

IABC Implantation With Primary St nting

The role of IABC in hemodynamically stable patients in the era of intracoronary stent implantation is at present unclear. A Dutch randomized, controlled study[41] involving 238 AMI patients followed for 3.5 years showed that treatment with counterpulsation did not significantly reduce death, nonfatal MI, stroke, or low LVEF at 6 months or enzymatic infarct size. On the other hand, a univariate analysis from a more recent retrospective study[42] of 1122

AMI patients who presented in cardiogenic shock demonstrated that IABC deployment was associated with a 35% reduction in the risk of death. A total of 28% of patients received counterpulsation in this population-based study.

Given the emerging trend toward adjunctive treatment in percutaneous coronary intervention (PCI), including the introduction of stenting and glycoprotein IIb/IIIa receptor blockade in the setting of PTCA, the adjunctive role of IABC for stent implantation in patients with AMI was evaluated. Among 63 patients who underwent primary stenting to the IRA, 10 developed cardiogenic shock to LV failure: 8 with LAD and 2 with right coronary artery lesions. The balloon pump was inserted after diagnostic catheterization in the cath lab, then patients were transferred for urgent cardiac surgery (primary stent implantation). This approach was successful in 9 out of the 10 patients.

Simultaneous Use of IABC in Thrombolytic Therapy: The TACTICS Experience

As mentioned previously, IABC may play a role as an adjunct to thrombolytic therapy because low BP (</= 85 mm Hg) diminishes the effectiveness of thrombolysis. The recent Thrombolysis And Counterpulsation To Improve Cardiogenic Shock Survival (TACTICS) trial assessed the effect of counterpulsation on patients with AMI who received TPA or streptokinase, together with intravenous heparin and aspirin. Balloons were inserted within 2 to 4 hours (median, 30 min) after thrombolysis onset and maintained at a 1:1 rate over 48 hours.

Although rates of diabetes, prior MI, and anterior MI were considerably higher in the 30 patients randomly allocated to thrombolysis plus IABC (*P* </= .09 for each comparison with the thrombolysis-alone group), individuals assigned to counterpulsation, particularly patients with severe hemodynamic deficits, exhibited modest clinical benefits, including a reduction in mortality that is consistent with observational studies using IABC in cardiogenic shock.

Prophylactic IABC for High-Risk Patients With Acute Myocardial Infarction

Coronary Artery Bypass Grafting

High-risk coronary patients consume a disproportionate share of available healthcare resources, including longer stays in the hospital and intensive-care unit (ICU). Counterpulsation may improve outcomes after revascularization by limiting the amount of ischemic myocardium. Independent risk factors for postoperative morbidity and mortality after CABG include prior CABG (ie, "redo" procedure), UA warranting surgical intervention on an urgent basis, low postoperative LVEF (/= 70%) left-main stenosis.

In prospective, randomized controlled trials, Dr. Christenson and colleagues[43,44] evaluated the influence of preoperative IABC in high-risk candidates for CABG. Nearly 90% of patients had LVEF

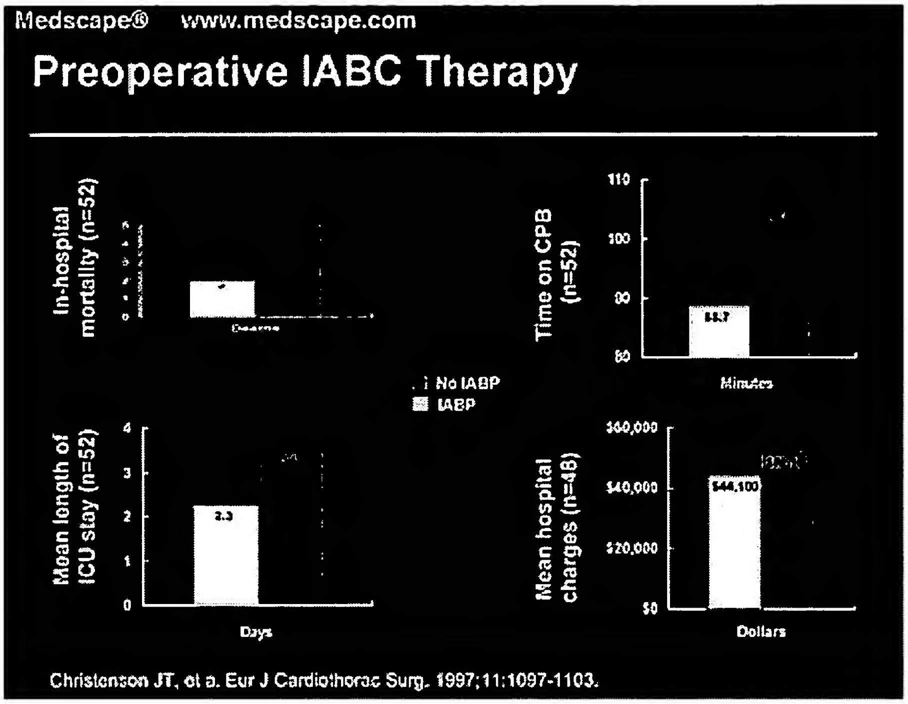

Figure 3. Effects of preoperative IABC on patients undergoing coronary artery bypass grafting.[43]

As shown in Figure 3, compared with their counterparts not treated with preoperative counterpulsation (controls), patients who received IABC prior to CABG exhibited significantly lower in-hospital mortality and morbidity. There were 2 fatalities in the IABC group (6%), as opposed to 5 (25%) in the control arm ($P = .049$), and only 6 patients in the IABC arm exhibited postoperative low cardiac output (19%), as opposed to 13 (60%) in the control group ($P = .046$).

Mean cardiac index values were significantly higher in the IABC group both before cardiopulmonary bypass and at 5 minutes to 24 hours after IABC. These clinical benefits sharply reduced hospital resource utilization. First, time on cardiopulmonary bypass was about 89 minutes in preoperative IABC patients as compared with 105.5 minutes in the control group ($P < .001$). Second, the mean length of ICU in patients treated with counterpulsation before IABC was 2.3 days, as compared with 3.5 days in the non-IABC -- a 34% decline ($P < .004$).

Although overall lengths of hospitalization were nonsignificantly lower in the IABC group (10.2 vs 14.9 days; $P = .078$), the mean total hospital charge among survivors was $44,100 in the IABC group as compared with $52,100 in the control group. In addition to shorter stays on ICUs, IABC patients required significantly less inotropic support with dobutamine and amrinone 6 to 24 hours after CABG.

A pooled analysis by Dr. Christenson's group[45] showed that total hospital costs were nearly 40% lower in high-risk CABG patients who underwent IABC before aortic cross-clamping ($27,780) than their counterparts who received no such circulatory support ($43,637). Finally, rates of GI and renal complications, including permanent renal failure and multiorgan failure, tended to be lower in the IABC group (consistent with enhanced cardiac output), although these differences were not statistically significant.

IABC facilitates posterior-vessel off-pump CABG in high-risk patients. Displacement of the heart during the procedure can decrease stroke volume, cardiac output, and BP, worsening regional myocardial ischemia; these adverse effects may be particularly serious in high-risk patients. Preoperative counterpulsation may also promote safe induction of anesthesia.

Among 142 high-risk patients with indications for IABC (eg, UA, significant left-main disease, intractable resting angina), surgical results in high-risk patients with preoperative or intraoperative IABC -- including ventilator-support time, ICU stay, and number of distal/posterior-vessel anastomoses -- were similar to those in lower-risk patients who did not receive perioperative counterpulsation.

PTCA

The value of instituting IABC prior to high-risk PTCA was supported in part by a large prospective registry reported by Brodie and coworkers.[46] Of 1490 consecutive patients with AMI treated with primary PTCA over a 13-year interval (238 high-risk patients), adverse cath lab events occurred in 5.9% of patients, including cardiopulmonary arrest in 3.1%, ventricular fibrillation in 4.0%, and protracted hypotension in 2%. Of all prognostic factors for cath-lab events, cardiogenic shock was the most robust (OR, 2.18), and low LVEF also increased the risk of adverse events by more than 2-fold.

Preinterventional IABC resulted in more than a 50% decline in cath-lab event rates among patients with cardiogenic shock: to 14.5% from 35.1% in patients who did not receive such treatment ($P = .009$). Counterpulsation also significantly reduced rates of cardiopulmonary arrest (6.5% vs 22.8%; $P = .01$) and ventricular fibrillation (12.9% vs 29.8.%; $P = .02$) in patients with cardiogenic shock. There were no cath-lab events in patients with CHF or low LVEF who were treated with IABC, whereas there were 15 in those who did not ($P = .10$). Finally, among all 238 high-risk patients, IABC significantly decreased overall event rates, as well as the occurrence of cardiopulmonary arrest and prolonged hypotension considered individually.[46]

Exploring the Possibilities of Permanent IABC

Powered pneumatically, the implantable IABC consists of the balloon pump, skin connector, and external drive unit. Permanent counterpulsation devices are sutured to the wall of the thoracic aorta in the same anatomic position as the traditional device. In preclinical experience, the device became pseudoendothelialized, essentially removing it from exposure to the circulation. The skin connector facilitated integration into animal tissues, with skin biopsies and fibroblast cultures evidencing wound healing within 2 weeks after implantation, and keratin production around the wound and good acceptance at 3 to 6 years thereafter.

In clinical trials, permanent implantable IABC exhibited the familiar hemodynamic benefits conferred by traditional counterpulsation devices, increasing cardiac output and decreasing both LV end-diastolic pressure and pulmonary capillary wedge pressure. The advantages of implantable IABC include nonobligatory use; the device can be turned on and off at the physician's discretion. In addition, the permanent device is nonthrombogenic, and many patients -- including those in heart failure -- can be maintained without inotropes. Ongoing challenges to development of a permanent implantable counterpulsation device include infectious complications and the prospect of leakage from the device.

Springboard to Pathophysiology: Are the Hemodynamic Benefits of IABC Attributable to Diastolic Augmentation or LV Unloading?

Diastolic Augmentation

In animal studies involving IABC, progressively more blood was retrieved as LA pressure increased to levels exceeding the colloidal osmotic pressure associated with pulmonary edema.

Together with LV bypass, counterpulsation introduces additional reductions in the ratio of myocardial oxygen consumption to CBF, which is on the order of 10:1 at baseline and increases with coronary-artery occlusion. Further, in part by decreasing LV wall tension, IABC might diminish ventricular irritability and thus represent a valuable approach to patients with intractable ventricular arrhythmias, including ventricular fibrillation (VFib), tachycardia, and incessant monomorphic ventricular tachycardia.[47,48]

Abolition of VFib depends in part on maintenance of coronary sinus pressure. Coronary arterial occlusion can alter myocardial K^+ flux, with the emergence of K^+ gradients at the ischemic border. Thus, both VFib and certain forms of conduction blocks can be abrogated if the coronary microvascular perfusion pressure is maintained above critical closing pressure (40 mm Hg). This ensures continued filtration of K^+ as well as adequate tissue oxygenation.

In a retrospective analysis[47] of ventricular arrhythmia seen in tertiary-care centers over a 5-year interval, 21 patients with comorbid LV impairment were treated with IABC. Of these cases, 18 were stabilized, and counterpulsation reduced the overall frequency of ventricular arrhythmias without eliciting IABC-related complications.

In addition, in a landmark trial conducted by Mueller and colleagues,[49] 24 patients in cardiogenic shock associated

with AMI were treated with either IABC, isoproterenol, or L-norepinephrine. Counterpulsation substantially enhanced regulation of myocardial metabolism and improved systemic perfusion. Both myocardial oxygen and lactate extraction approached normal levels with IABC; this form of circulatory support also increased the cardiac index, CBF, and mean aortic pressure by 0.45 L/min/m^2, 23 mL/100 g/min, and 15 mm Hg, respectively.

LV Unloading: Influenc of Arterial Compliance

On the other hand, 2 clinical trials reported in 1998 suggested that the hemodynamic effects conferred by counterpulsation can also be ascribed to a marked decline in aortic impedance. Using a validated technique developed at the Hippokration Hospital in Athens, Stefanadis and colleagues[50] measured aortic diameters with an intravascular catheter and aortic pressure with a catheter-tip micromanometer.

Among 12 patients in cardiogenic shock, counterpulsation enhanced aortic distensibility and diminished peripheral arterial wave reflection. Treatment with IABC significantly increased both aortic distensibility and the cardiac index (by 30% and 24%), as well as reducing myocardial O_2 consumption by 31% ($P < .001$). In addition, both improvements in cardiac index and myocardial energy balance were significantly related to aortic stiffness, suggesting that the hemodynamic benefits of IABC were due to enhanced aortic elasticity (ie, reduced afterload).

Marchionni's group[51] attributed increases in LV stroke work to a decrease in arterial elastance (Ea) associated with counterpulsation. This parameter can be estimated from the end-systolic pressure:stroke volume ratio. Among 18 patients with complicated AMI or UA, IABC significantly augmented stroke index while significantly reducing aortic end-systolic pressure, Ea, systemic vascular resistance, mean right atrial pressure, and mean pulmonary artery wedge pressure. Maximal benefits were observed with a 40-mL (vs 20-mL) balloon volume. These investigators concluded that "the increase in LV stroke work was closely related to the decrease in Ea." Thus, cardiogenic shock patients with relatively high Ea values at presentation may be more likely to benefit from IABC than individuals with lower Ea.

In patients with cardiogenic shock after AMI, arterial compliance has been measured by a noninvasive method: applanation tonometry -- using pulse-wave analysis of the radial artery. In a clinical trial, counterpulsation decreased systolic aortic pressure (SAP) by 8.8% and end-diastolic arterial pressure by 7.8%. A higher aortic impedance (stiffness) was associated with higher diastolic augmentation indices; augmentation index values in excess of 100% predicted improved IABC performance.

Effects of Balloon Parameters on Hemodynamic Performance of IABC: A Mathematical Mod l

The need and rationale for the development of a mathematical model of circulatory function were predicated in part on the fact that the aortic-pressure waveform differs for each beat and each patient. The mathematical model formulated by Dr. Ohley's group[52] incorporated the following variables: fluid mass density; coefficient of viscosity; Young's modulus; proportionality coefficient for artery stretch; wall thickness; aortic-section radius; various hemodynamic parameters (eg, HR, SBP, CO); as well as a number of balloon-operation properties, such as timing, location, diameter, and volume.

IABC in Pediatric Patients

The physiologic rationale for IABC in infants and children is fundamentally similar to that in adults, namely, to provide support for the failing heart. Mechanical circulatory support devices can enhance myocardial recovery and survival, particularly in the face of technically difficult procedures and anticipated difficulty weaning from the heart-lung machine.

Veasy and colleagues at the University of Utah[53-55] reported clinical benefits of IABC in children with low cardiac output refractory to pharmacologic management. In a retrospective analysis involving 29 children treated with IABC -- mainly for repair of congenital defects -- from 1981 to 1990, balloons were placed surgically through an end-to-side graft to the femoral artery in children aged 1 week to 13 years. Survival rates tended to be higher with advancing age, but the data were not inconsistent with use in infants; indeed, the youngest survivor was only 1 month old (body weight < 10 lb). Maximal duration of counterpulsation reached 7 days. In cases seen at this institution since 1994, the long-term survival is 82%.

Other forms of pediatric circulatory support include extracorporeal membrane oxygenation (ECMO), CBP, and pneumatic LV-assist devices (LVAD).[56] Early and long-term survival following ECMO (mean duration 109 hours) were 50% and 43% in a study at the Children's Hospital of Pittsburgh.[57] Most of these children had either postcardiotomy contractile dysfunction or dilated cardiomyopathy. Although 8 of the 14 ECMO-treated patients required surgical decompression for pulmonary edema, a total of 9 (64%) went on to receive cardiac transplants. Sepsis and bleeding complications, as well as labor intensiveness, are among several current challenges that must be overcome before ECMO becomes a reliable bridge to transplantation. Other pediatric treatment options include pulsatile-flow paracorporeal LVADs, which are not available in the United States.

Conclusions

For interventional cardiologists confronted with low-output cardiogenic shock, which can severely compromise prognosis in the setting of AMI, UA, heart failure, and other cardiovascular diseases, intra-aortic balloon pumping represents a sound clinical option. Contemporary trends toward more frequent percutaneous placement in the cath lab on a proactive basis, rather than in the operating theater as a "last-ditch" response to cardiogenic shock, reflect the numerous potential hemodynamic and clinical benefits associated with counterpulsation, particularly among high-risk patients undergoing PCI or cardiotomy. Counterpulsation remains the mechanical circulatory support modality of first choice.

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Authors and Disclosures

Authors

Spyridon D. Moulopoulos, MD, PhD, ScD

Professor Emeritus, Department of Clinical Therapeutics, University of Athens Medical School; Director, Department of Clinical Therapeutics, Alexandra Hospital, Athens, Greece. Dr. Moulopoulos is an Honorary Member of the French and Greek Societies of Cardiology, and a Corresponding Member of the French Medical Academy

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